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 NEWS  1           Web Page URLs for STN Seminar Schedule - N. America
 NEWS  2           "Ask CAS" for self-help around the clock
 NEWS  3  FEB 27   New STN AnaVist pricing effective March 1, 2006
 NEWS  4  APR 04   STN AnaVist $500 visualization usage credit offered
 NEWS  5  MAY 10   CA/CAplus enhanced with 1900-1906 U.S. patent records
 NEWS  6  MAY 11   KOREAPAT updates resume
 NEWS  7  MAY 19   Derwent World Patents Index to be reloaded and enhanced
 NEWS  8  MAY 30   IPC 8 Rolled-up Core codes added to CA/CAplus and
                   USPATFULL/USPAT2
 NEWS  9  MAY 30   The F-Term thesaurus is now available in CA/CAplus
 NEWS 10  JUN 02   The first reclassification of IPC codes now complete in
                   INPADOC
 NEWS 11  JUN 26   TULSA/TULSA2 reloaded and enhanced with new search and
                   and display fields
 NEWS 12  JUN 28   Price changes in full-text patent databases EPFULL and PCTFULL
 NEWS 13  JUl 11   CHEMSAFE reloaded and enhanced
 NEWS 14  JUl 14   FSTA enhanced with Japanese patents
 NEWS 15  JUl 19   Coverage of Research Disclosure reinstated in DWPI
 NEWS 16  AUG 09   INSPEC enhanced with 1898-1968 archive
 NEWS 17  AUG 28   ADISCTI Reloaded and Enhanced
 NEWS 18  AUG 30   CA(SM)/CAplus(SM) Austrian patent law changes

 NEWS EXPRESS  JUNE 30 CURRENT WINDOWS VERSION IS V8.01b, CURRENT
               MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),
               AND CURRENT DISCOVER FILE IS DATED 26 JUNE 2006.

 NEWS HOURS    STN Operating Hours Plus Help Desk Availability
 NEWS LOGIN    Welcome Banner and News Items
 NEWS IPC8     For general information regarding STN implementation of IPC 8
 NEWS X25      X.25 communication option no longer available

Enter NEWS followed by the item number or name to see news on that
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FILE 'HOME' ENTERED AT 13:39:33 ON 31 AUG 2006

=> file caplus
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                                                       ENTRY    SESSION
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```
=> s somatostatin or neurotensin or penetratine or bombensin
         19356 SOMATOSTATIN
           146 SOMATOSTATINS
         19365 SOMATOSTATIN
                 (SOMATOSTATIN OR SOMATOSTATINS)
          4752 NEUROTENSIN
            27 NEUROTENSINS
          4755 NEUROTENSIN
                 (NEUROTENSIN OR NEUROTENSINS)
             0 PENETRATINE
             1 PENETRATINES
             1 PENETRATINE
                 (PENETRATINE OR PENETRATINES)
             1 BOMBENSIN
L1       23282 SOMATOSTATIN OR NEUROTENSIN OR PENETRATINE OR BOMBENSIN

=> s acridine or porphyrin or ellipticine or phenantroline or carbazole or
benzimidazole or daunorubicine or epirubicine or mixoxantrone
         17981 ACRIDINE
          1711 ACRIDINES
         18378 ACRIDINE
                 (ACRIDINE OR ACRIDINES)
         35685 PORPHYRIN
         24812 PORPHYRINS
         41961 PORPHYRIN
                 (PORPHYRIN OR PORPHYRINS)
          1033 ELLIPTICINE
           147 ELLIPTICINES
          1057 ELLIPTICINE
                 (ELLIPTICINE OR ELLIPTICINES)
           171 PHENANTROLINE
             5 PHENANTROLINES
           174 PHENANTROLINE
                 (PHENANTROLINE OR PHENANTROLINES)
         16646 CARBAZOLE
          2183 CARBAZOLES
         17214 CARBAZOLE
                 (CARBAZOLE OR CARBAZOLES)
         23371 BENZIMIDAZOLE
          5898 BENZIMIDAZOLES
         24718 BENZIMIDAZOLE
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                (BENZIMIDAZOLE OR BENZIMIDAZOLES)
            42 DAUNORUBICINE
            16 EPIRUBICINE
             0 MIXOXANTRONE
L2      102010 ACRIDINE OR PORPHYRIN OR ELLIPTICINE OR PHENANTROLINE OR CARBAZO
               LE OR BENZIMIDAZOLE OR DAUNORUBICINE OR EPIRUBICINE OR MIXOXANTR
               ONE

=> s l2 and l2
L3      102010 L2 AND L2

=> s l2 and l1
L4          53 L2 AND L1

=> s conjugat? or coupl? or link? or combin?
        225632 CONJUGAT?
        783227 COUPL?
        466608 LINK?
       1115681 COMBIN?
L5     2438342 CONJUGAT? OR COUPL? OR LINK? OR COMBIN?

=> s l5 and l4
L6          29 L5 AND L4

=> s l6 not py>1999
       7078308 PY>1999
L7            1 L6 NOT PY>1999

=> d ibib

L7   ANSWER 1 OF 1  CAPLUS  COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER:        1989:625888  CAPLUS
DOCUMENT NUMBER:         111:225888
TITLE:                   Enprostil reduces the increase of gastric corpus
                         mucosal mass induced by the hydrogen-potassium-
                         stimulated adenosine triphosphatase inhibitor BY
                         831-78 in the rat
AUTHOR(S):               Inauen, W.; Rohner, C.; Koelz, H. R.; Herdmann, J.;
                         Schuerer-Maly, C. C.; Varga, L.; Halter, F.
CORPORATE SOURCE:        Gastrointest. Unit, Univ. Hosp., Bern, 3010, Switz.
SOURCE:                  Gastroenterology (1989), 97(4), 846-52
                         CODEN: GASTAB; ISSN: 0016-5085
DOCUMENT TYPE:           Journal
LANGUAGE:                English


=> d abs kwic

L7   ANSWER 1 OF 1  CAPLUS  COPYRIGHT 2006 ACS on STN
AB   It was determined if enprostil, a synthetic PGE2 derivative, might inhibit
gastrin
     release and the trophic effects on gastric oxyntic mucosa induced by
     prolonged treatment with an inhibitor of H+-K+-stimulated ATPase, the
     substituted benzimidazole BY 831-78.  Rats were treated
     intragastrically with enprostil (1 or 15 μg/kg b.i.d.), BY 831-78 (15
     μmol/kg once daily), the  combination of enprostil and BY
     831-78, ranitidine (300 μmol/kg b.i.d.), and placebo.  Plasma gastrin
     and somatostatin levels and gastric acid secretion were measured
     during a 1-day treatment in animals fitted with chronic gastric fistulas
     and repeatedly during 9 wk of treatment in intact rats.  Despite
     inhibiting acid secretion, enprostil did not increase plasma gastrin.
     When combined with BY 831-78, enprostil transiently reduced the
     BY 831-78-induced increase of integrated plasma gastrin (1375 vs. 2137
     pmol/L.12h) in fasted rats with fistulas, but failed to prevent the marked
     hypergastrinemia following 9 wk of treatment with BY 831-78 (717 vs. 731
```

pmol/L) in intact rats. However, enprostil reduced the BY 831-78-induced increase of oxyntic mucosal volume (458 vs. 567 mm3), whereas BY 831-78 prevented the enprostil-induced increase of antral mucosal volume (42 vs. 56 mm3). Apparently, some of the trophic effects induced by a H+,K+-ATPase inhibitor are not exclusively governed by gastrin.

AB . . . and the trophic effects on gastric oxyntic mucosa induced by prolonged treatment with an inhibitor of H+-K+-stimulated ATPase, the substituted benzimidazole BY 831-78. Rats were treated intragastrically with enprostil (1 or 15 μg/kg b.i.d.), BY 831-78 (15 μmol/kg once daily), the combination of enprostil and BY 831-78, ranitidine (300 μmol/kg b.i.d.), and placebo. Plasma gastrin and somatostatin levels and gastric acid secretion were measured during a 1-day treatment in animals fitted with chronic gastric fistulas and repeatedly during 9 wk of treatment in intact rats. Despite inhibiting acid secretion, enprostil did not increase plasma gastrin. When combined with BY 831-78, enprostil transiently reduced the BY 831-78-induced increase of integrated plasma gastrin (1375 vs. 2137 pmol/L.12h) in fasted. . .

IT 51110-01-1, Somatostatin
RL: BIOL (Biological study)
(secretion of, ATPase inhibitor and PGE2 analog effect on, gastrin in relation to)

```
=> s l6 not py>2000
      6188416 PY>2000
L8            2 L6 NOT PY>2000

=> s l8 not l7
L9            1 L8 NOT L7

=> d ibib abs kwic

L9   ANSWER 1 OF 1  CAPLUS  COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER:         2000:690483  CAPLUS
DOCUMENT NUMBER:          133:361093
TITLE:                    Ligand-induced internalization of neurotensin
                          in transfected COS-7 cells: differential intracellular
                          trafficking of ligand and receptor
AUTHOR(S):                Vandenbulcke, Franck; Nouel, Dominique; Vincent,
                          Jean-Pierre; Mazella, Jean; Beaudet, Alain
CORPORATE SOURCE:         Montreal Neurological Institute, McGill University,
                          Montreal, QC, H2A 2B4, Can.
SOURCE:                   Journal of Cell Science (2000), 113(17), 2963-2975
                          CODEN: JNCSAI; ISSN: 0021-9533
PUBLISHER:                Company of Biologists Ltd.
DOCUMENT TYPE:            Journal
LANGUAGE:                 English
```

AB The neuropeptide neurotensin (NT) is known to be internalized in a receptor-mediated fashion into its target cells. To gain insight into the mechanisms underlying this process, we monitored in parallel the migration of the NT1 neurotensin receptor subtype and a fluorescent analog of NT (fluo-NT) in COS-7 cells transfected with a tagged NT1 construct. Fluo-NT internalization was prevented by hypertonic sucrose, potassium depletion and cytosol acidification, demonstrating that it proceeded via clathrin-coated pits. Within 0-30 min, fluo-NT accumulated together with its receptor in Acridine Orange-pos., acidic organelles. These organelles concentrated transferrin and immunostained pos. for rab 5A, therefore they were early endosomes. After 30-45 min, the ligand and its receptor no longer colocalized. Fluo-NT was first found in rab 7-pos. late endosomes and later in a nonacidic juxtanuclear compartment identified as the Trans-Golgi Network (TGN) by virtue of its staining for syntaxin 6. This juxtanuclear compartment also stained pos. for rab 7 and for the TGN/pericentriolar recycling endosome marker rab 11, suggesting that the ligand could have been recruited to the TGN from

either late or recycling endosomes. By that time, internalized receptors were detected in Lamp-1-immunoreactive lysosomes. These results demonstrate that neurotensin/NT1 receptor complexes follow a recycling cycle that is unique among the G protein-coupled receptors studied to date, and provide the first evidence for the targeting of a nonendogenous protein from endosomes to the TGN.

REFERENCE COUNT: 73 THERE ARE 73 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

TI Ligand-induced internalization of neurotensin in transfected COS-7 cells: differential intracellular trafficking of ligand and receptor

AB The neuropeptide neurotensin (NT) is known to be internalized in a receptor-mediated fashion into its target cells. To gain insight into the mechanisms underlying this process, we monitored in parallel the migration of the NT1 neurotensin receptor subtype and a fluorescent analog of NT (fluo-NT) in COS-7 cells transfected with a tagged NT1 construct. Fluo-NT internalization was prevented by hypertonic sucrose, potassium depletion and cytosol acidification, demonstrating that it proceeded via clathrin-coated pits. Within 0-30 min, fluo-NT accumulated together with its receptor in Acridine Orange-pos., acidic organelles. These organelles concentrated transferrin and immunostained pos. for rab 5A, therefore they were early endosomes. After 30-45 min, the ligand and its receptor no longer colocalized. Fluo-NT was first found in rab 7-pos. late endosomes and later in a nonacidic juxtanuclear compartment identified as the Trans-Golgi Network (TGN) by virtue of its staining for syntaxin 6. This juxtanuclear compartment also stained pos. for rab 7 and for the TGN/pericentriolar recycling endosome marker rab 11, suggesting that the ligand could have been recruited to the TGN from either late or recycling endosomes. By that time, internalized receptors were detected in Lamp-1-immunoreactive lysosomes. These results demonstrate that neurotensin/NT1 receptor complexes follow a recycling cycle that is unique among the G protein-coupled receptors studied to date, and provide the first evidence for the targeting of a nonendogenous protein from endosomes to the TGN.

ST neurotensin complex NT1 receptor endocytosis intracellular trafficking

IT Organelle
(coated pit; neurotensin internalization via NT1 receptors proceeds via clathrin-coated pits)

IT Endosome
(internalized neurotensin/NT1 receptor complexes are initially targeted to endosomes upon import)

IT Biological transport
(intracellular; neurotensin internalized via NT1 receptors is recruited to trans-golgi network whereas receptors are targeted to lysosomes for degradation)

IT Lysosome
(neurotensin internalized via NT1 receptors is recruited to trans-golgi network whereas receptors are targeted to lysosomes for degradation)

IT Neurotensin receptors
RL: BOC (Biological occurrence); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence); PROC (Process)
(neurotensin internalized via NT1 receptors is recruited to trans-golgi network whereas receptors are targeted to lysosomes for degradation)

IT Endocytosis
(receptor-mediated; neurotensin internalization via NT1 receptors proceeds via clathrin-coated pits)

IT Organelle
(trans-Golgi network; neurotensin internalized via NT1 receptors is recruited to trans-golgi network whereas receptors are targeted to lysosomes for degradation)

IT 39379-15-2, Neurotensin
RL: BOC (Biological occurrence); BPR (Biological process); BSU (Biological

study, unclassified); BIOL (Biological study); OCCU (Occurrence); PROC (Process)
(neurotensin internalized via NT1 receptors is recruited to trans-golgi network whereas receptors are targeted to lysosomes for degradation)

=>

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